

# **EXHIBIT 28**

**UNITED STATES DISTRICT COURT  
DISTRICT OF NEW JERSEY**

**IN RE JOHNSON & JOHNSON  
TALCUM POWDER PRODUCTS  
MARKETING, SALES PRACTICES,  
AND PRODUCTS LIABILITY  
LITIGATION**

**MDL NO. 16-2738 (FLW) (LHG)**

***THIS DOCUMENT RELATES TO ALL CASES***

**RULE 26 EXPERT REPORT OF  
ARCH CARSON, MD, PHD**

Date: November 16, 2018

A handwritten signature in blue ink, appearing to read "Arch Carson MD", is written over a horizontal line.

Arch Carson, MD, PhD

## **Talcum Powder and Ovarian Cancer**

### **1. Introduction**

I was asked to explain the relationship between the regular perineal use of talc-based personal hygiene products and the subsequent development of ovarian cancer in their users. I intend this report to explain this relationship. I will describe ovarian cancer, what is known about its natural history, and will present statistics regarding its incidence, prevalence and fatality. I will then describe what talc is and why talcum powder is used in personal care products. I will then present the scientific evidence linking talc-based personal hygiene products and their components with cancer, and will show how the various components of this evidence, along with other data, lead me to conclude that regular perineal application of talcum powder products causes ovarian cancer in some users, and raises the risk of ovarian cancer in all users.

### **2. Qualifications**

I am a physician who specializes in the practice of medical toxicology. I am currently an Associate Professor at the University of Texas School of Public Health in Houston and the Program Director of the Occupational and Environmental Medicine Residency training program at the University of Texas Health Science Center at Houston. I received my medical degree from the Ohio State University and a doctor of philosophy degree in Toxicology from the Kettering Laboratory at the University of Cincinnati. I am board certified by the American Board of Preventive Medicine in Occupational Medicine, and have been in the continuous practice of medical toxicology since 1991. My professional activities have included patient care, basic and applied research, teaching of medical students, graduate students and post-graduate medical trainees, and professional consulting. I have been a program director of the NIOSH-funded Education and Research Center at the University of Texas for 19 of the last 21 years. Other major collaborations include as Liaison for the World Health Organization Collaborating Centre in Occupational Health and as environmental exposure consultant to the MD Anderson Cancer Center in Houston. My curriculum vitae is attached to this report as Exhibit A.

### **3. Information reviewed and methodology employed**

In the preparation of this report, I have reviewed relevant published scientific and medical literature, reports and documents produced in the process of litigation, and various other documents and websites that I believed to be pertinent to the refinement or extension of my professional opinions. I applied the same methodology and scientific rigor in this research that I use in my academic and clinical practice. Documents and other sources which I considered in reaching my opinions are listed in Exhibit B, "Materials and Data Considered."

### **4. What is ovarian cancer?**

#### **a. What is cancer?**

All types of cancer involve the uncontrolled growth and accumulation or dissemination of cells that originated from normal cells, but have been altered so that they behave differently. The many cells of a single cancer that result from this change are typically all derived from a single progenitor cell, and represent a clone of cells. When this clone

reaches sufficient numbers, the cells themselves may develop into a recognizable “mass” that is called a tumor. Tumors may cause symptoms and other health problems simply by taking up space and putting pressure on neighboring structures or blocking important fluid channels or nerves, thus disrupting normal functions of the body. Still other cancers can proliferate into the blood stream. As the number of cancerous cells increase, the biochemically active substances that they produce can also become a problem resulting in abnormal biological responses throughout the body. Some substances that might become a problem in this way include normal or abnormal hormones, enzymes, antibodies, and proteins. Cancerous cells are considered malignant if they lose their normal tendency to stop proliferating when they have filled a space or the bounds of their particular tissue type, referred to as contact inhibition. Malignant cells ignore these boundary cues and may invade other tissue spaces and organs with devastating results. They may also migrate via the blood stream or other routes to distant sites within the body where they set up a new location of tumor growth and tissue invasion. This process is called metastasis. Typically, cancers are not diagnosed until they produce sufficient symptoms or biochemical abnormalities that lead to an exhaustive diagnostic search resulting in their discovery. Occasionally, cancers are discovered accidentally as part of another investigation, e.g. a chest x-ray may find an asymptomatic lung cancer; a blood test may disclose a telltale abnormality. Still fewer cancers are discovered before they cause health problems through screening tests that are sensitive and specific enough to detect common cancers at a preclinical and hopefully highly treatable stage, e.g. routine colonoscopies to detect colon cancer, or PSA blood tests to detect prostate cancer.

b. Carcinogenesis-a two-step process

The process of normal cells becoming cancer cells is generally recognized as resulting from a two-step process.

**Initiation.** During initiation, a change is produced at one or more places in the DNA of a cell’s chromosomes. Because the DNA represents the genetic code that becomes duplicated and passed along to cells that arise from it, when that cell divides to produce two cells, the change to the genetic code is also duplicated and is present in both of them.

Normally, the abnormal cell that results from a change in the genetic code cannot survive because its cellular machinery is also abnormal and poorly or non-functional. Less often, if the cell is able to survive in the body, it is still abnormal and deformed, and is recognized by the body’s immune system as alien. The immune system attacks it and destroys it, and it does not survive. In the very rare instance that an alteration to the genetic material results in a survivable hereditary change that is not fatal, and which can escape the surveillance of the body’s immune system, the resulting clone may live and persist. (Coussens LM, 2002)

**Promotion** - Once a cancer clone has been produced, it is at risk for being discovered and destroyed by the body’s immune system, or failing to thrive in an environment for which it is not suited. Promotion is the process by which the cancer clone is shielded

from the body's defenses and is stimulated to undergo rapid growth, transforming a microscopic cancer clone into a self-sustaining symptomatic cancer over time. (Ferrante D, 2007) (Coussens LM, 2002)

Most known carcinogenesis events occur by the two-step process and involve a long latent period between the moment of the alteration in the genetic material and the recognition that a cancer is present. In human cancers, this latent period is often several months to many years in length. The latency period for ovarian cancer, generally, and for cancers induced by environmental agents is usually quite long, often >20 years. (Nadler DL, 2014) Promotion occurs throughout the latent period and stimulates the growing cancerous cells to become a recognizable cancer. A third stage in the natural history of a cancer, referred to as Progression, involves maturation, differentiation or de-differentiation and accumulation of transcriptional changes that solidify the tumor's growth rate and invasiveness. Some carcinogenic substances are initiators and some are promoters, and still others are called complete carcinogens because they are capable of initiation and promotion.

c. Ovarian cancer

Ovarian cancer is a group of cancers that arise in the ovary or in adjacent tissues. It is estimated that about 22,240 women will receive a new diagnosis of ovarian cancer and about 14,070 women will die from ovarian cancer in the United States in 2018. (American Cancer Society, n.d.) (Torre LA, 2018) Ovarian cancer ranks fifth in cancer deaths among women, and first due to cancers of the female reproductive system. Most ovarian cancers are not discovered until they have reached an advanced stage and have spread to sites elsewhere in the body. Because advanced ovarian cancers are more difficult to treat, they have a high fatality rate. For these reasons, any effective prevention of ovarian cancer or reduction in ovarian cancer risk can have a significant impact on this disease and can save many women's lives.

There are several recognized forms of ovarian cancer that are distinguished by the specific tissues from which they arise, or the microscopic characteristics of the tumor cells themselves. About 85% to 90% of malignant ovarian cancers are epithelial ovarian carcinomas, and the majority of these are of the serous type (American Cancer Society, n.d.) (Prat, 2015). Ovarian, fallopian tube, and peritoneal cancers have a similar clinical presentation and are treated similarly, and current evidence suggests that they may have a common origin, supporting a common staging system (Soong TR, 2018).

Despite significant advances in cancer diagnosis and therapies over the past several decades, there have been few changes in the incidence or fatality rates for ovarian cancer. Consequently, it is worth considering preventable environmental causes of the ovarian cancer epidemic. (Woodruff, 1979) (LA Torre, 2018)

5. What is talc?

a. General

Talc is a hydrated magnesium silicate mineral produced through a metamorphic geological process and having the generalized chemical formula  $\text{Mg}_3\text{Si}_4\text{O}_{10}(\text{OH})_2$ . Some substitution of atoms occurs in variations of talc found in nature. Small amounts of Aluminum (Al) or Titanium (Ti) can substitute for Silicon, and small amounts of Iron (Fe), Manganese (Mn), Aluminum (Al) and Calcium (Ca) can substitute for Magnesium. This produces slight variations in the color, hardness and chemical properties of the mineral. Talc is the softest mineral on the Mohs Hardness Scale. (King, n.d.) It is essentially insoluble in water, but is slightly soluble in dilute mineral acids. The process seems to involve the extraction of magnesium and other cations leaving only the silicate as silicic acid and silica.

The commercial value of talc stems from its crystalline structure. Most talc is present in natural deposits as the platy form of talc, in which the talc crystals are arranged in large flat sheets running parallel to one another. These sheets are attracted to each other by weak Van der Waals forces that can be easily overcome by mechanical forces, causing the sheets to slide on each other. On the macro scale, this property gives talc its characteristic slippery feeling on the skin. The platy structure also gives talc its ability to absorb moisture and oil. Some talc is found as a fibrous crystalline structure, similar to some asbestos, also a magnesium silicate mineral. In fact, these two minerals are closely related in terms of their formation and composition. Talc deposits are often intermingled with asbestos and vice versa. (Rohl, 1974) (Rohl AN, 1976) (National Institute for Occupational Safety and Health, 2011) (Lockey, 1981)

**b. Talcum Powder and Cancer.**

Numerous studies have examined the cancer causing characteristics of talc. (Wild, 2006) Talc has caused cancer when implanted in various tissues and under the skin in laboratory animals. It causes inflammation and fibrotic reaction, including the chemotaxis of inflammatory immune cells, and accelerated growth and division of cells in the involved tissues (Okada, 2007). This is a normal body process that leads to the thwarting of infection and rapid healing, but in the absence of tissue injury, accelerated growth and cell division has the effect of amplifying and propagating viable genetic mutations, leading to cancer. Talc particles have been repeatedly demonstrated in ovarian tumor tissues (Henderson WJ C. J., 1971) (Henderson WJ T. H., 1979) and in inflammatory tissue in otherwise normal ovaries (Mostafa SAM, 1985). In 2006, the International Agency for Research on Cancer (IARC) evaluated the published evidence for the carcinogenicity of talc, not containing asbestiform fibers, when inhaled into the respiratory system and when applied to the perineum in personal hygiene activities. The agency concluded that talcum powder is a “possible human carcinogen” (Group 2B) when applied to the perineum, meaning that there is insufficient evidence of carcinogenesis in humans, but strong evidence in other mammalian species. IARC also concluded that there was insufficient evidence of carcinogenicity by the inhalation route (Group 3). (International Agency for Research on Cancer, 2010) Since that time,

numerous other studies have added to the data on this issue. A recent meta-analysis showed that talc workers do have an excess of lung cancers. (Chang C-J, 2017)

When implanted under the skin or into tissues of laboratory animals, talcum powder induces an inflammatory response. This reaction involves the chemotaxis of inflammatory cells of the immune system, lymphocytes, neutrophils and macrophages, the release of cytokines that promote membrane permeability and stimulate cell division. As this reaction matures over time, granulomas may begin to develop. All of this signifies that talcum powder is an effective and potent promotor of already initiated genetic alterations. (Fletcher NM M. I., 2018) (Fletcher NM S. G., 2018) (Saed GM, 2017) (Radić I, 1988) (Okada, 2007) Other studies have demonstrated the ability of these same reactions to satisfy the carcinogenic initiation step, characterizing talcum powder as a complete carcinogen. (Shukla A, 2009) (Fletcher NM M. I., 2018)

c. What about asbestos and other components in talc and talc-based products?

Talcum powder products in the marketplace have been shown to contain asbestos. (Paoletti L, 1984) (VanOrden D, 2000) (VanGosen BS, 2004) (Longo WE, 2017) Asbestos is conclusively recognized as a cause of ovarian cancers. The IARC Working Group concluded that “a causal association between exposure to asbestos and cancer of the ovary was clearly established, based on five strongly positive cohort mortality studies of women with heavy occupational exposure to asbestos, (International Agency for Research on Cancer, 2012)” and “studies showing that women and girls with environmental, but not occupational exposure to asbestos had positive, though non-significant, increases in both ovarian cancer incidence and mortality. (Acheson ED, 1982) (Fox, 1982) (Berry G, 2000) (Newhouse ML, 1972) (Reid A H. J., 2008) (Reid A S. A., 2009) (Pira E, 2005) (Magnani C, 2008) (Bertolotti M, 2008) (Ferrante D, 2007) (Germani D, 1999) (Rösler JA, 1994) The classification determined by IARC included all forms of asbestos and talc containing asbestiform fibers (fibrous talc). I have seen evidence that Johnson & Johnson’s talcum powder products contain asbestos and fibrous talc.<sup>1</sup>

d. Carcinogenic metals in talcum powder

In addition to other related minerals, talcum powder may contain varying amounts of chromium, cobalt and nickel, metal ions that are recognized as cancer causing. These ions leach out of the talcum powder slowly over time, resulting in continuous, low-level exposure of the surrounding tissues to carcinogenic metals. (Jurinski JB, 2001) I have seen evidence that Johnson & Johnson’s talcum powder products contain nickel (Group 1

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<sup>1</sup> Ex. 28, Hopkins Dep. (Aug. 16 & 17, 2018; Oct. 26, 2018; and Nov. 5, 2018); Ex. 47, Pier Dep. (Sept. 12 & 13, 2018); Expert Report of William E. Longo, PhD and Mark W. Rigler, PhD (Nov. 14, 2018)

human carcinogen), chromium (Group 1 human carcinogen), and cobalt (Group 2B-possible human carcinogen).<sup>2</sup>

e. Other potentially cancer-causing constituents

Johnson & Johnson's Baby Powder and Shower to Shower contain numerous ingredients that have been added to the products, i.e. fragrance chemicals, some of which have been shown to produce cancer in laboratory animals. These substances are likely to be present in very small or trace quantities, and likely present a lower level of risk than the major components, by mass. Nonetheless, any additional risks are added as part of a total risk profile. I have reviewed the report of Dr. Michael Crowley and agree with his conclusions that these chemicals may contribute to the inflammatory properties, toxicity, and potential carcinogenicity of the products.<sup>3</sup>

6. Epidemiology linking talcum powder and ovarian cancer

Many research studies have shown a strong association between talcum powder exposure and the development of ovarian cancer. (Langseth H, 2008) (Terry KL, 2013) (Schildkraut JM, 2016) (Trabert, 2016) (Berge W, 2017) (Cramer Daniel W, 2016) (Penninkilampi R, 2018)

a. What evidence links exposure to talcum powder products with ovarian cancer?

Multiple epidemiological studies have examined the link between the personal hygiene use of talc containing products and the occurrence of ovarian cancers (Booth M, 1989) (Cook LS K. M., 1997) (Cook LS e. a., 1997) (Cramer DW, 1982) (Whittemore AS, 1988) (Harlow BL W. B., 1989) (Chen Y, 1992) (Harlow BL C. D., 1992) (Rosenblatt KA, 1992) (Hartge P, 1988) (Tzonou A, 1993) (Chang S, 1997) (Heller DS, 1996) (Penninkilampi R, 2018). Talcum powder causes proliferation of human (Prat, 2015) ovarian cells in culture (Buz'Zard AR, 2007), and causes these cells to express reactive oxygen species (ROS) (Buz'Zard AR, 2007).

The research investigating the link between talcum powder exposure and ovarian cancer has been reviewed as a scientific whole at multiple stages. (Harlow BL H. P., 1995) (Ness Roberta B, 1999) (Muscat JE, 2008) (Terry KL, 2013) (Berge W, 2017) (Penninkilampi R, 2018)

Laboratory, animal and human studies support the conclusions that talc causes ovarian cancer, and have filled in the blanks that establish biological plausibility and scientific coherence. (Jaiswal M, 2000) (Balkwill Fran, 2001) (Okada, 2007) (Saed Ghassan M, 2017) (Harper, 2019)

7. Talcum powder product use

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<sup>2</sup> Ex. 47, Pier Dep. (Sept. 12 & 13, 2018)

<sup>3</sup> Expert Report of Michael Crowley, PhD (Nov. 12, 2018).



Numerous studies have interviewed women regarding their personal practices of application of talc-based powders to the perineal area. Due to variations in these practices, it has been difficult to estimate dose in order to evaluate the dose response relationship for ovarian cancer. It is also difficult to exactly estimate the quantity of talcum powder administration during personal hygiene activities. For studies that attempted to determine amount of exposure, most relied on a method of estimating the frequency of application and/or the duration of those practices, then simply multiplying to reach a total number of applications over time. (Harlow BL H. P., 1995) (Langseth H, 2008) A review of studies of perineal talcum powder or cornstarch application suggests that the use of cornstarch instead of talcum powder reduces the risk of ovarian cancer. (Whysner J, 2000)

#### 8. Other evidence

- a. Transport of talc-containing materials from the perineum to the upper reproductive tract and body cavities has been shown to occur with startling regularity and with respect to a wide variety of particulate materials. (Egli GE, 1961) (Venter PF, 1979) (Blumenkrantz MJ, 1981) (Halme J, 1984) (Sjösten ACE, 2004) Clearly, sufficient particulate materials applied routinely to the perineum have ready access and in sufficient quantities to produce biological responses in internal tissues, including the ovaries and surrounding structures. There are a limited number of animal studies suggesting that this transport does not occur. (National Toxicology Program, 1993) These are not as compelling as the human evidence because of anatomical and physiological differences between animals and humans in this regard, as well as the overwhelming evidence in humans.

#### 9. Conclusions and opinions

The following conclusions and opinions are expressed with respect to reasonable medical and scientific certainty and I have applied reliable scientific principles and methods to the facts in reaching them. These opinions are based upon the documents and literature reviewed and cited herein, and also upon my own professional training and experience in practice of medicine and medical toxicology.

##### **I. Talcum powder products sold for personal hygiene use are carcinogenic.**

Talcum powder is immunogenic, producing chronic inflammation in the tissues in which it sequesters, with the attraction of lymphocytes and macrophages and the ongoing local release of pro-inflammatory cytokines and reactive oxygen species. Further, all talcum powder has some component of mineral fibers that are toxic to macrophages and intensify the inflammatory response and stimulate cell growth and proliferation. The presence of asbestos, fibrous talc, carcinogenic metals and other chemicals further intensify this effect. Cohort and case-control studies have shown statistically significant associations between talc-based powder use and ovarian cancers. The presence of carcinogenic metals such as, chromium, cobalt and nickel, and toxic fragrance components in commercial talcum powder products, adds to their carcinogenic potency. Talcum powder is a complete carcinogen and can both initiate and promote the development of cancers in the tissues in which it sequesters.

## **II. Perineal use of talcum powder products for feminine hygiene purposes results in direct exposure to the female reproductive tract.**

A proportion of talcum powder from personal hygiene applications to the perineum is transported or migrates through the reproductive tract, through the patent fallopian tubes, onto the ovaries and into the pelvic cavity. Talc particles have been identified in reproductive system structures of women who utilize talc powders. These include the uterine cervix, the endometrium, the fallopian tubes and the ovaries. Inhalation is likely a secondary route of exposure.

## **III. Common carcinogenic constituents of talcum powder products participate in and add to the carcinogenic process.**

Naturally occurring carcinogenic components of talcum powder, i.e. asbestos, chromium, nickel, and cobalt, are liberated in bodily fluids and tissues and are free to exert their carcinogenic effects. Added substances that are toxic or carcinogenic, i.e. fragrance chemicals, may also contribute to these effects. This process is the most intense where the duration is the longest. Because the ovaries have no intrinsic elimination system, the transport of talc particles and their constituents reaches the ovaries where it stalls and sequesters. For these reasons, ovarian tissue is most at risk for the carcinogenic effect of these substances.

## **IV. Regular perineal application of talcum powder products causes epithelial ovarian cancer in some users, and raises the risk of ovarian cancer in all users.**

Multiple case-control and cohort epidemiological studies have looked at the relationship between the perineal use of talc-based powders and the eventual development of epithelial ovarian cancer. Most, but not all, of these studies show a consistent positive relationship. When confounding and bias are exhaustively considered, the positive association remains. I conclude that the apparent cause and effect relationship between perineal talcum powder use and ovarian cancer is real, amounting to about a 30% increased risk of ovarian cancer in talcum powder product users. At the current rate of ovarian cancer diagnosis and mortality, elimination of this source of risk could result in over 3,000 lives saved in the U.S. each year.

In 1965, Sir Austin Bradford Hill published what has come to be recognized as the best collection of factors to consider for the assessment of scientific evidence that relates the causation of disease to environmental exposures (Hill, 1965). These factors include: (1) Strength of association, (2) Consistency of the evidence, (3) Specificity, (4) Temporality, (5) Biological gradient, (6) Plausibility, (7) Coherence, (8) Experiment, and (9) Analogy. Below I provide my evaluation of the scientific evidence with respect to the Hill factors.

**Strength of association** –Many epidemiological studies have attempted to examine the association between perineal use of talcum powder products and ovarian cancer. Most of these have been case-control studies, where women diagnosed with ovarian cancer are paired with others of similar demographic background who do not have ovarian cancer. All of these women are interviewed about their past practices and exposures, including the use of talcum powder products. The resulting data are analyzed to compute an odds ratio (OR) that describes the

likelihood of those with cancer having had greater exposure to talcum powder than those who did not. Cohort studies selected populations of women, assessing them for many factors, including perineal talcum powder use, and followed them over time counting the occurrences of ovarian cancers. These studies were then able to compute a relative risk (RR) of exposure to talcum powder resulting in ovarian cancers. Of more than 25 case-control studies in the literature, the heavy majority showed positive and significant ORs for perineal talcum powder use and ovarian cancer. The three cohort studies did not find a significant relative risk of perineal talcum powder exposure leading to ovarian cancer, but did show positive non-significant trends. Several research groups have looked at the totality of the research evidence, evaluated the published study reports, and have reanalyzed those data on a common playing field through meta-analyses. Taken in their totality, and accounting for sources of bias and differing statistical treatments, these epidemiological studies support a strong association between the perineal use of talcum powder and ovarian cancer.

**Consistency of the evidence** – As stated above, the majority of epidemiological studies that have investigated the link between perineal talcum powder use and ovarian cancer have reported positive associations. These studies are consistent in their findings of a relationship between perineal use of talcum powder products and the development of ovarian cancer. Further, recent meta-analyses of previously published studies have verified the comparability of the research methods used and the consensus of conclusions.

**Specificity** – Specificity is the concept that a specific disease, rather than a host of diseases, is produced by a particular exposure, and that the exposure is a principal cause of the disease. Although talcum powder is known to cause non-specific inflammation in many tissues where its residues locate, the stimulation of ovarian cancer is particularly associated with the presence of talc in the ovaries and fallopian tubes. Of known factors associated with ovarian cancer, i.e. nulliparous state, early menarche, late menopause, oral contraceptive use, living in the twentieth century and beyond, perineal talcum powder exposure is proving to be prominent among them.

**Temporality** – If a particular exposure is the cause of a particular disease, then the onset of exposure should precede the onset of the disease. Studies investigating the link between perineal talcum powder exposure and ovarian cancer are designed to compare those with prior exposure to those who are not exposed, and so the scientific evidence supports this consideration.

**Biological gradient** – A basic toxicological principle is that a greater exposure intensity will result in a larger proportion of those exposed expressing the toxic effect, in this case ovarian cancer. In order to determine the intensity of a long-term environmental exposure, typically a measure of frequency or quantity of use is multiplied by the duration of such use. This allows categorization of exposure levels and comparisons. Although some studies have failed to find evidence of a dose-response relationship, several more recent reports have shown a clear dose-response when the number of subjects rose to a level producing sufficient statistical power to allow the analysis after subdivision of subjects into pertinent categorical groups, and frequency and duration were measured (Schildkraut JM, 2016) (Cramer Daniel W, 2016) (Wu, et al., 2009).

**Plausibility** – This factor expects the rational presentation of a mechanism whereby the exposure in question leads to the disease. Thus, if no such mechanism can be proposed, it is less likely that causation will be supported. In the case of ovarian cancer, the mechanism supported in the literature is as follows: Talcum powder products are applied to the perineal area in the course of routine personal hygiene practices. This element is supported by the existence of these products in the marketplace for many years and the statements of subjects interviewed for the purpose of conducting the scientific research discussed elsewhere in this report. Portions of the applied powders are transferred via active processes or passive mass action movements into the female reproductive tract, some making it all the way to the distal fallopian tubes, the ovary surfaces and the pelvic and peritoneal cavities. This element is supported by the observations that particulate materials of differing variety can make their ways along these pathways to the listed destinations, and the finding and confirmation of talc particles in normal ovarian tissues and ovarian tumor tissues at the time of oophorectomy or autopsy. Once reaching the target tissues, talcum powder and its constituents initiate carcinogenesis via multiple means, including, inflammation with chemotaxis of inflammatory cells, liberation of cytokines, and reactive oxygen species, inactivation of TP53 genetic modulator, inhibition of DNA repair, and long-term promotion of genetic mutations via continuous inflammation and cellular growth stimulation.

**Coherence** – The proposed cause and effect relationship should not “seriously conflict with the generally known facts of the natural history and biology of the disease.”(Hill, 1965) The proposal that talcum powder product use results in the occurrence of ovarian cancer is entirely consistent with what is known about other factors related to ovarian cancer, i.e. early menarche, late menopause, pregnancies, breastfeeding history, oral contraceptive use, etc. All are factors that influence the local inflammatory environment of the ovary and its surroundings and have the potential to promote existing transcriptional errors and mutations.

**Experiment** – Interventions, such as tubal ligation that decreases the incidence of ovarian cancer by blocking the exposure route, offers experimental support for this mechanism. The use of cornstarch-based dusting powders as a substitute for talcum powder products offers additional experimental support.

**Analogy** – Have there been other environmental exposures that have been associated with ovarian cancers that act via similar mechanisms? Talcum powder is somewhat unique in terms of its delivery mechanism. But beyond that, the case of asbestos exposure is similar. Asbestos exposure has resulted in excesses of ovarian cancers in exposed women, although the route of exposure is thought to be by inhalation. Nonetheless, asbestos is a mineral very similar both chemically and structurally to talc that has been found in the ovary and peritoneal cavity of exposed women. The mechanisms of carcinogenesis for both asbestos and talc are similar and analogous. Further, talc-based products contain asbestos and non-asbestos mineral fibers having carcinogenic potential.

When considering these factors, I gave the most weight to the compelling strength of association and consistency, as well as the well-described biologic mechanism.

The currently available scientific research, when considered in its totality, demonstrates a cause and effect relationship between the use of talcum powder products and the development of epithelial ovarian cancer. This opinion is reinforced by my consideration of the Hill factors for the assessment of causation.

In reviewing the scientific and medical literature on talcum powder product use, I also performed a risk assessment and considered whether perineal use of those products poses a safety risk to consumers. This involved careful consideration of the epidemiological literature, data on the dose-response relationship and exposure, as well as the nature of these products, which are used primarily for personal care. I also considered evidence of the toxicity of these products, for which repeated testing and analyses have shown to contain carcinogens.

In considering the weight of this epidemiologic, toxicologic, and mechanistic evidence, across multiple studies, time, demographics, and researchers, demonstrating a consistent association between perineal use of talcum powder products and ovarian cancer, it is my opinion that talcum powder products increase the risk of ovarian cancer and pose a significant health hazard.

In conclusion, it is my opinion that the perineal use of talcum powder products causes ovarian cancer in some users and increases the risk of ovarian cancer in all users of these products.

All of my opinions in this report are provided with respect to a reasonable degree of medical and scientific certainty. I reserve the right to amend or supplement my report as new information becomes available.

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# Exhibit A

## Curriculum Vitae

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### Biosketch

Arch "Chip" Carson, MD, PhD is a physician (The Ohio State University), board certified in Occupational Medicine (American Board of Preventive Medicine), who holds a Doctor of Philosophy degree in Toxicology (University of Cincinnati, Kettering Laboratory). He has served on the faculty of the University of Cincinnati and the New York University Medical Center and joined the faculty of the University of Texas School of Public Health in 1992 in its Environmental Sciences Discipline and Occupational and Environmental Health and Aerospace Medicine Module. He is Associate Professor of Occupational Health, directs the Occupational and Environmental Medicine Residency Program and is a member of the research team of the Southwest Center for Occupational and Environmental Health, a NIOSH Education and Research Center, and WHO Collaborating Centre in Occupational Health. He maintains a clinical practice of occupational medicine and medical toxicology. In his more recent role as Medical Director for the University of Texas Medical Branch in Galveston, he is responsible for the health monitoring and care of more than 15,000 employees. He is a frequent consultant to governments, corporations and the legal community on matters related to industrial chemical exposure, toxicology and environmental justice. His research interests include: environmental and occupational chemical exposures, inhalation injuries, metal exposures and cancer, and professional training in occupational medicine.

### Professional Activities/Employment

2017-18	University of Texas Medical Branch, Galveston, Assistant Clinical Professor of Preventive Medicine and Family Medicine
2017-18	University of Texas Medical Branch, Galveston, Medical Director, Employee Health Services.
2017-18	Enbridge Corporation, Houston Texas, Medical Director, Employee Health Services.
2010-18	University of Texas Health Science Center, Houston, Associate Professor of Occupational Health.
2010-18	University of Texas Health Science Center at Houston, Program Director, Occupational and Environmental Medicine Residency.
1991-18	Private practice of Occupational Medicine and Toxicology, New York, Texas and Ohio.
2011-18	Spectra Energy Corporation, Houston Texas, Medical Director, Employee Health Services.
1997-13	Texas Medical Center Inc., Houston Texas, Medical Director, Employee Health Services.
1992-08	University of Texas School of Public Health, Assistant Professor of Occupational Medicine and Environmental Sciences.
1998-08	University of Texas Health Science Center at Houston, Program Director, Occupational and Environmental Medicine Residency.
2003-08	Southwest Center for Occupational and Environmental Health, Principal Investigator and Director, Diller Phosgene Exposure Incident Registry of the American Chemistry Council.

2000-06 Chevron Phillips Chemical Company, Houston Texas, Corporate Medical Director.

2003-05 U.S. Department of Energy Office of Worker Advocacy Physician Review Panel Appointee.

1997-04 Southwest Center for Occupational and Environmental Health, Principal Investigator, City of Houston Lead Poisoning Epidemiology Project.

1992-03 UT Health Services, University of Texas Houston Health Science Center, Attending Physician, Occupational Medicine and Toxicology.

1997-01 University of Houston Downtown, Medical Director, Student Health Service.

1998-99 University of Texas School of Public Health, Convener of the Occupational/Environmental Health and Aerospace Medicine Module.

1992-97 Respiratory Consultants of Houston, PA, Attending Physician, Occupational Medicine and Toxicology.

1992-95 Exxon Chemical Americas, Baytown Polymer Center and Basic Chemicals Technology, Baytown TX, Consultant Physician.

1990-91 New York University Medical Center, Bellevue Hospital, Tisch Hospital, and Manhattan VA Hospital, New York NY, Dept. of Medicine, Clinical Instructor.

1982-90 Chemical Information Services Inc, Cincinnati OH, Associate in Toxicology.

1978-87 University of Cincinnati College of Medicine, Cincinnati OH, Instructor and Lecturer, Adjunct Assistant Professor of Industrial Toxicology.

1974-79 University of Cincinnati College of Medicine, Kettering Laboratory, Cincinnati OH, Research Technologist in Occupational Medicine and Clinical Studies.

1969-74 Millstone Inc., Cincinnati OH, Design Engineer, environmental control systems.

#### **Educational Background**

2002 Certificate of Board Eligibility, Medical Toxicology, American Board of Preventive Medicine/American Board of Emergency Medicine

1992 Certificate of Training - Residency in Occupational Medicine University of Texas Health Science Center at Houston, School of Public Health, and Southwest Center for Occupational and Environmental Health, Houston TX, 1992.

1991 Certificate of Training - Postgraduate Internship in Internal Medicine, New York University Medical Center and Bellevue Hospital Center, New York NY.

1990 MD - Ohio State University College of Medicine, Columbus OH.

1987 PhD - Kettering Laboratory, University of Cincinnati College of Medicine, Cincinnati OH, awarded in the field of "Environmental Health – Toxicology."

1973 BS - University of Cincinnati College of Arts and Sciences Cincinnati OH. Awarded in "Biological Sciences with Concentration in Engineering."

1969 Rensselaer Polytechnic Institute, Troy NY. Management Engineering

1968 Villa Madonna College, Covington KY. Certificate in Contemporary Physics.

#### **Fellowships**

2011-13 UTHHealth, Health Educators Fellowship, University of Texas Health Science Center at Houston.

- 1983-85 American Lung Association Fellowship in Lung Research (Inhalation Toxicology), American Lung Association of Southwestern Ohio, Grant.
- 1981-82 Owens Corning Fiberglas, Graduate Research Fellowship in Combustion Toxicology.
- 1979-80 National Institute for Occupational Safety and Health, Centers for Disease Control, Doctoral Fellowship in Industrial Toxicology.

#### **Certifications**

- 2012 License to practice medicine, State of Ohio 35.098635
- 2010 Certified Healthy Homes Specialist – National Environmental Health Association.
- 2002 Board Eligibility, Medical Toxicology, American Board of Preventive Medicine/American Board of Emergency Medicine.
- 1994 Board Certification, Occupational Medicine, American Board of Preventive Medicine.
- 1992 License to practice medicine, State of Texas J2524.
- 1991 License to practice medicine, State of New York 186563.
- 1982 Emergency Hazard Response, Environmental and Industrial Chemical Accident Management, U.S. Environmental Protection Agency.
- 1979 Pulmonary Function Testing for Occupational Surveillance, NIOSH #003.

#### **Professional Community Service**

- 2013-18 University of Texas Health Science Center at Houston, Steering Committee on Interprofessional Collaboration
- 2013-18 University of Texas Health Science Center at Houston, Chemical Safety Committee.
- 1998-18 Association of Environmental and Occupational Clinics/ATSDR community resource on toxic exposures and health consequences, Federal Region VI.
- 1997-18 City of Houston Biological, Chemical and Radiation Emergency Preparedness Program. Medical Toxicology On-Call Advisor to the Houston Medical Strike Team.
- 1998-18 Association of Occupational and Environmental Medicine Residency Directors. Chairman 2005-2006
- 2010-18 University of Texas Health Science Center at Houston, Graduate Medical Education Committee
- 1997-08
- 2010-18 University of Texas Health Science Center, Houston, Community/Press Resource and Speaker via Public Information Office, (Toxic Exposures and Environmental Health).
- 1994-08
- 1996-18 American College of Occupational and Environmental Medicine, Council on Academic Affairs and Co-chair, Academic Section 2004-2006. Occupational Medicine Residency Directors Committee, Chair 2006-2007, Appointed Member, Taskforce on the Future of Occupational Medicine Education 2005-2007. Appointed Co-chair, Taskforce on the Future of Occupational Medicine Education 2013-2015.
- 1996-18 Texas College of Occupational and Environmental Medicine. Secretary/Treasurer-2004-5, President Elect-2005-6, President-2006-7, Past President 2007-8.
- 2003-12 Boy Scouts of America, Sam Houston Council, Registered Adult Leader and Merit Badge Counselor.
- 2005-08 University of Texas School of Public Health, Practice Council Co-chair

2003-05	U.S. Department of Energy Office of Worker Advocacy Physician Review Panel Appointee.
1996-00	American Public Health Association, Occupational Health Subcommittee
1994-96	Advisory Board, National Environmental Education and Training Center (NEETC), Curriculum Development Committee.
1981-85	Tri-State Air Committee Inc., Cincinnati OH, (voluntary air quality organization) Scientific Advisor, Elected to Board of Directors in 1982, President and Chairman 1984-85.
1981-85	American Lung Association of Southwestern Ohio, Cincinnati OH, (voluntary health organization) speakers bureau.
1982-83	City of Cincinnati, Appointment to Occupational Health Scientific Liaison Board (municipal advisory committee).
1981-83	Cincinnati Area Toxic Substances Coalition, Cincinnati OH, (coalition of business, voluntary, and labor organizations with interest in environmental toxic substance issues) Cofounder and Chairman.
1982-83	Ohio River Valley Committee on Occupational Safety and Health, Cincinnati OH, (organized labor coalition) Scientific Resource Committee.
1972-82	Walnut Hills-Evanston Medical Center, Cincinnati OH, (primary care center) Board of Directors.

#### Professional Societies

1991-18	American College of Occupational and Environmental Medicine.
1991-18	Texas College of Occupational and Environmental Medicine
2007-18	Texas Public Health Association.
2006-18	International Congress on Occupational Health.
2003-18	American College of Medical Toxicology.
2002-06	Society of Occupational and Environmental Health.
2001-06	American Conference of Governmental Industrial Hygienists.
1994-00	American Public Health Association.
1983-87	American Industrial Hygiene Association.
1983-87	Society of Toxicology.
1980-85	American Thoracic Society, Associate Member and Participant in Occupational and Environment Scientific Session.

#### Publications

Anderson F, **Carson A**, Whitehead L and Burau K. Age, Race and Gender Spatiotemporal Disparities of COPD Emergency Room Visits in Houston, Texas. Occupational Diseases and Environmental Medicine. 3:1-9, 2015. <http://dx.doi.org/10.4236/odem.2015.31001>.

Anderson F, **Carson A**, Whitehead L and Burau K. Spatiotemporal Analysis of the Effect of Ozone and Fine Particulate on CVD Emergency Room Visits in Harris County, Texas. Open Journal of Air Pollution, 3:87-99, 2014. <http://dx.doi.org/10.4236/ojap.2014.34009>.



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Delclos GL, Tullis LA, **Carson A**. The services industry. In *Occupational and Environmental Lung Diseases*. Tarlo SM, Cullinan, Nemery B eds. 2010 pp.258-271. Wiley-Blackwell, West Sussex, UK.

Pugach S, Clarkson T, (**Carson A**). Prenatal mercury exposure and postnatal outcome: clinical case report and analysis. *Clin Toxicol* 47:366-370, 2009.

Pauluhn J, **Carson A**, Costa DL, Gordon T, Kodavanti U, Last JA, Matthay MA, Pinkerton KE and Sciuto AM. Workshop summary: phosgene-induced pulmonary toxicity revisited: appraisal of early and late markers of pulmonary injury from animal models with emphasis on human significance. *Inhalation Toxicology*. 19(10):789-810, 2007.

Delclos GL, Gimeno D, Arif AA, Burau KD, **Carson A**, Lusk C, Stock T, Symanski E, Whitehead LW, Zock JP, Benavides FG and Anto JM. Occupational risk factors and asthma among health care professionals, *American Journal of Respiratory & Critical Care Medicine*. 175(7):667-75, 2007.

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Delclos GL, Arif AA, Aday L, **Carson A**, Lai D, Lusk C, Stock T, Symanski E, Whitehead LW, Benavides FG and Anto JM. Validation of an asthma questionnaire for use in healthcare workers. *Occupational & Environmental Medicine*. 63(3):173-9, 2006.

Delclos GL, Bright KA, **Carson A**, Felknor SA, Mackey TA, Morandi MT, Schulze LJH and Whitehead LW. "A global survey of occupational health competencies and curriculum" *International Journal of Occupational and Environmental Health*; 11:181-194, 2005.

Nooka A, Duonghi L, **Carson A**, Hassan M. Assessing Occupational Risk for Pancreatic Cancer by Chemical Exposures and Work History: A Case-Control study at MD Anderson Cancer Center. American Association for Cancer Research, Orlando. March, 2004.

Mitchell CS, Moline J, Avery AN, Baker D, Blessman JE, **Carson A**, Cosby O, Darcey D, Ducatman A, Emmett EA, Forst L, Gerr F, Gochfeld M, Guidotti TL, Harber P, Hu H, Hegmann KT, Kipen HM, Levin J, McGrail MP, Meyer JD, Mueller KL, Prince S, Rubin R, Schwerha JJ, Sprince NL, Taiwo O and Upfal M. In response to the 2002, vol. 22, no. 4 article entitled "The rise and fall of occupational medicine in the United States" [Letter] *Am J Preventive Med*. 23(4):307-9, 2002.

**Carson A** and Delclos GL. "The Respiratory System," in *Modern Industrial Hygiene: Volume II – Biological Aspects*, JL Perkins, ed. 2003, American Conference of Governmental Industrial Hygienists, Cincinnati.

**Carson A**, Colombo S and Alavi. A, City of Houston Childhood Lead Poisoning Prevention Program: Case Density and Impact Analysis, March 31, 2000, Technical Report (Principal Investigator).

Townsend MC, Lockett JE, Velez H, **Carson A**, Cowl CT, Delclos GL, Gerstenhaber BJ, Harber PI, Horvath EP, Jolly AT, Jones SH, Knackmuhs GG, Lindesmith LA, Markham TN, Raymond LW, Rosenberg DM, Sherson D, Smith DD, and Wintermeyer SF. ACOEM Position Statement – "Spirometry in the Occupational Setting" *JOEM*; 42: 228-245, 2000.

Bright K, Delclos G, **Carson A**, Felknor S, Mackey T, Morandi M, Schultz L and Whitehead L. A Global Study of Occupational Health Competencies and Curricula, Report to the World Health Organization, March, 2000, Southwest Center for Occupational and Environmental Health.



**Carson A**, Guevara E, Delclos GL, Murray KA, Burau KD, Morandi MT, Felknor SA, ("A Study of General Health of Workers of the Industrial Complex of Barrancabermeja") in [Compendium on Occupational Health in the Petroleum Industry of Colombia: Technical and Scientific Report of the "Occupational Health in the Petroleum Industry" Project], 1999 Pan American Health Organization (co-author).

**Carson A**, Hangoc V and Bahrainwala M, City of Houston Childhood Lead Poisoning Prevention Program: Case Density and Impact Analysis, June 30, 1999, Technical Report (Principal Investigator).

**Carson A**, Spears B, and Burau K, City of Houston Childhood Lead Poisoning Prevention Program: Case Density and Impact Analysis, June 30, 1998, Technical Report (Principal Investigator).

**Carson A**, Detry M, Spears B, and Burau K, City of Houston Childhood Lead Poisoning Prevention Program: Case Density and Impact Analysis, June 30, 1997, Technical Report (Principal Investigator).

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**Carson A**, "Respiratory Effects of Exposure to Fresh Smokes from Pyrolytic Decomposition of Styrene Plastic in Rats." Doctoral Dissertation, University of Cincinnati Kettering Laboratory, 1987.

**Carson A**, "A Dynamic Method for the Generation of Fresh Smokes From Combustion or Pyrolytic Decomposition of Structural Materials." Doctoral Dissertation, University of Cincinnati Kettering Laboratory, 1987.

Samuels SJ, Lemasters GK and **Carson A**, "Statistical Methods for Describing Occupational Exposure Measurements," Am. Ind. Hyg. Assoc. J., 46:427-433, 1985.

Lemasters GK, **Carson A** and Samuels SJ, "Occupational Styrene Exposure for Twelve Product Categories in the Reinforced-Plastics Industry," Am. Ind. Hyg. Assoc. J., 46:434-441, 1985.

Lockey JE, Brooks SM, Jarabek AM, Khoury PR, McKay RT, **Carson A**, Morrison JA, Wiot JF and Spitz HB. "Pulmonary changes after exposure to vermiculite contaminated with fibrous tremolite" Am Rev Respir Dis. 129(6):952-8, 1984.

Lockey JE, Jarabek A, **Carson A**, McKay R, Harber P, Khoury P, Morrison J, Wiot J, Spitz H and Brooks SM, "Pulmonary Hazards from Vermiculite," in Health Issues Related to Metal and Nonmetallic Mining, WL Wagner, W Rom and P Merchant eds. 1983, Butterworth's, Boston.

Vinegar A and **Carson A**, "Pulmonary Function Changes in Chinese Hamsters Exposed Six Months to Diesel Exhaust," Environ Int, 5:369-371, 1981.

Lockey JE, Jarabek A, **Carson A**, McKay R, Harber P, Khoury P, Morrison J, Prior J and Brooks SM, "Health Effects of Vermiculite Exposure," Am Rev Respir Dis, 123:133, 1981 abstract.

Lockey JE, Jarabek A, **Carson A**, McKay R, Harber P, Khoury P, Morrison J and Brooks SM, "Single-Breath Diffusing Capacity (DLCOsb) in a Working Population," Am Rev Respir Dis, 123:132, 1981 abstract.

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**Carson A**, Vinegar A, Leng J and Cooper G, "Effects of Chronic Exposure to some Diesel Exhaust Components on Lung Function in Rats," Fed Proc, 39:1091, 1980 abstract.

Elia VJ, Anderson LA, MacDonald TJ, **Carson A**, Buncher CR and Brooks SM, "Determination of Urinary Mandelic and Phenylglyoxylic Acids in Styrene Exposed Workers and a Control Population," Am Ind Hyg Assoc J, 41:922-926, 1980.

Brooks SM, Anderson LA, Emmett E, **Carson A**, Tsay JY, Elia VJ, Buncher CR and Karbowsky R, "The Effects of Protective Equipment on Styrene Exposure in Workers in the Reinforced Plastics Industry," Arch Environ Health, 35:287-294, 1980.

Brooks SM, Zipp T, Barber M and **Carson A**, "Measurement of Maximal Expiratory Flow Rates in Cigarette Smokers Using Gases of High and Low Densities," Am Rev Respir Dis, 118:75-81, 1978.

# Exhibit B

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- “A Survey of the Long-Term Effects of Talc and Kaolin Pleurodesis.” *British Journal of Diseases of the Chest* 73 (1979): 285–88.
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## DEPOSITIONS, TRANSCRIPTS AND REPORTS:

Affidavit of Laura Plunkett, PhD 02.22.18

Deposition of Alice Blount in the Ingham v. J&J Matter on 04.13.18

Deposition of Annie Awanaiss Yessian on 07.13.2017



Deposition and Exhibits of Pat Downey Dated 8.7.18-8.8.18  
Deposition and Exhibits of John Hopkins Dated 8.16.18-8.17.18, 10.17.18 and 11.05.18  
Deposition and Exhibits of Susan Nicholson Dated 7.26.18-7.27.18  
Deposition and Exhibits of Julie Pier Dated 9.12.18-9.13.18  
Ingham v. J&J Volume 11 (Egilman, Koman, Martinez, Packard) 6-14-18  
Ingham v. J&J Volume 14A (Madigan, Williams) 6-20-18  
Ingham v. JJ Volume 24A (Warner Huh, MD) 7.5.18  
Ingham v. JJ Volume 24B (Warner Huh, MD) 7.5.18  
John J. Godleski Expert Report for Brower Matter Dated 6.23.18  
Lanzo Plaintiffs MIL re Imerys Spoliation and Concealment of Talc Samples  
Laura Plunkett - Supplemental Expert Brower Report  
Longo Analysis of J&J's Historical Talc Samples from the 1960's  
Longo Analysis of J&J's Historical Talc Samples from the 1970's  
Longo Analysis of J&J's Historical Talc Samples from the 1980's  
Longo Analysis of J&J's Historical Talc Samples from the 1990's  
Longo Analysis of J&J's Baby Powder Historical Samples - Asian - October 2018  
Longo Analysis of J&J's BP Talc Products for Amphibole (Tremolite) Asbestos 8.2.17  
Longo Analysis Report\_Exhibit BB\_04.28.2017  
Longo MAS Project 14-1852 Below the Waist Application of Johnson's BP 9.2017  
Longo Process Blanks for the Analysis of J&J's Products from the 60's to 90's for Asbestos  
Longo TEM Analysis of Historical 1978 Johnson's BP Sample for Amphibole Asbestos 2.16.18  
Longo Verification of Lee Poye's TEM Analysis of J&J's Historical Vermont Talc 11.5.18  
Michael Crowley Expert Report Dated 11.12.18  
Report of Results: MVA11730 Investigation of Italian Talc Samples for Asbestos 08.01.2017  
RJLEE-001497  
Thomas Dydek Brower Expert Report Dated 8.16.18 (corrected on 8.20.18)  
Thomas Dydek Educational Report\_FINAL (4-9-2018)  
Thomas Dydek MDL Educational Report Dated 4.9.18

## **OTHER SOURCES:**

American Cancer Society Ovarian Cancer Statistics  
ATSDR Toxicological Profile for Asbestos  
EPA Chemical Assessment Summary for Asbestos - 2017  
EPA Guidelines for Carcinogen Risk Assessment - March 2005  
EPA Health Assessment Document for Talc - 1992  
Exhibit 1 - ATTORNEYS' EYES ONLY  
Exhibit 2 - ATTORNEYS' EYES ONLY  
Exhibit 3 - ATTORNEYS' EYES ONLY  
FDA 4-1-2014 Response Letter to Epstein Denying Petition  
Fitzgerald Analysis of J&J Baby Powder #1 and #2 Dated July 26, 2017  
IARC Monograph 100C - Arsenic, Metals, Fibres, and Dusts - Excerpts  
IARC Monograph 14 - Asbestos - 1977

IARC Monograph 2 - Some Inorganic and Organometallic Compounds - 1973

IARC Monograph 68 - Silica, Some Silicates, Coal Dust and Para-Aramid Fibrils - 1997

IARC Monograph 74 - Surgical Implants and Other Foreign Bodies - 1999

IARC Monograph 82 - Some Traditional Herbal Medicines, Some Mycotoxins, Naphthalene and Styrene - 2002

IARC Monograph 86 - Cobalt in Hard Minerals and Cobalt Sulfate, Gallium Arsenide, Indium Phosphide and Vanadium Pentoxide - 2006

IARC Monograph 87 - Inorganic and Organic Lead Compounds – 2006

IMERYS013188	J&J History
IMERYS045182	J&J S2s and BP Product Analysis - 1972
IMERYS045184	JNJ 000087928
IMERYS048311	JNJ 000088570
IMERYS051370	JNJ 000285351
IMERYS053387	JNJ000025132
IMERYS090653	JNJ000062359
IMERYS098115	JNJ000062436
IMERYS105215	JNJ000063608
IMERYS210136	JNJ000063951
IMERYS210729	JNJ000064544
IMERYS219720	JNJ000064762; JNJ000265171
IMERYS286445	JNJ000065264
IMERYS304036	JNJ000065601
IMERYS340454	JNJ000087710
IMERYS340798	JNJ000087716
IMERYS342524	JNJ000089413
IMERYS406170	JNJ000231304
IMERYS422289	JNJ000237076
IMERYS 088907	JNJ000237379
IMERYS 284935	JNJ000239723
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IMERYS209971	JNJ000245002
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IMERYS281335	JNJ000347962
IMERYS281776	JNJ000521616
IMERYS324700	JNJ000000704
IMERYS-A_0011817	JNJ000011150
IMERYS-A_0015663	JNJ000016645

JNJ000019415

JNJ000025132

JNJ000026987

JNJ000046293

JNJ000245678

JNJ000245762

JNJ000251888

JNJ000260700

JNJ000261010

JNJ000265536

JNJ000279507

JNJ000348778

JNJ000404860

PCPC\_MDL00062175

Pltf\_MISC\_00000272 (JANSSEN-000001-19)

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NIOSH Preliminary Report on Fiber Exposure During Use of Baby Powders - 1972

NTP Technical Report on the Toxicology and Carcinogenesis Studies of Talc (CAS No. 14807-96-6)- 1993

NTP Toxicology and Carcinogenesis Studies of Talc in F344/N Rats and B6C3F Mice Report No. 421

P-468

Read-the-Letter-from-the-FDA-on-Cosmetics

The Birth of Our Baby Products \_ Kilmer House

WCD 002478 - Exhibit 32 Waldstreicher

JNJ000460665

JNJ000526750

JNJ000886067

JNJAZ55\_000000577

JNJAZ55\_000000905

JNJAZ55\_000004563

JNJAZ55\_000008177

JNJL61\_000014431

JNJMX68\_000003728

JNJMX68\_000012858

JNJMX68\_000013019

JNJNL61\_000079334

**Arch Carson, MD, PhD Legal Testimony, 2015-2018**

Elaine Hale and Kenneth Dorsey parker, Jr. v. Centerpoint Energy Houston Electric, LLC; in the 55<sup>th</sup> District Court of Harris County, Texas.

2016

Harris County, TX

for Plaintiff

Danny Henderson and Linda Henderson; Magdaleno Flores and Maria Flores; Shari Waldrop; and Bryan Thomas v. Magnablend, Inc., Nugreen Specialty, Inc., Nugreen Solutions, Inc., and Enviro Tech Inc.; in the 40<sup>th</sup> District Court of Ellis County, Texas.

2015

Ellis County, TX

for Defendant

Edgar Guadalupe Solis v. Eastman Chemical Company, Texas Operations, Tradebe Environmental Services, Inc. d/b/a Tradebe Industrial Services LLC; in the 234<sup>th</sup> District Court of Harris County, Texas.

2015

Harris County, TX

for Defendant

Arch I. Carson, MD, PhD  
Professional Consultation Fee Schedule

Evidence-base research, report preparation, documentation, conference	\$450/hr
Interview, physical examination or medical testing of patients	450/hr
Review of documents	450/hr
Testimony at deposition or trial plus expenses	450/hr
Inspection, examination or sampling of physical evidence or sites	450/hr
Travel (Travel maximum \$4,000 per diem, plus expenses)	200/hr
Laboratory analyses/studies	at cost
Overhead and Supplies	at cost